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Influence of untreated periodontitis on osseointegration of dental implants in a beagle dog model

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이 대 현

- **Abstract –**

Influence of untreated periodontitis on osseointegration of dental implants in a beagle dog model

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Background: There have been previous studies on the relationship between periodontitis and periimplantitis, but limited information is available on how periodontitis affects osseointegration and wound healing of newly placed dental implants adjacent to natural teeth. The objective of the present experiment was to evaluate healing around dental implants adjacent to teeth with untreated experimental periodontitis.

Methods: The experiment included 6 male beagle dogs. Scaling and plaque control procedures were performed in 3 dogs (the control group). In the other 3 dogs (the

experimental group), retraction cords and ligature wires were placed subgingivally around all premolars and the first molars. Induced experimental periodontitis was confirmed after 3 months. Each control or experimental group was divided into 2 subgroups depending on the timing of implant placement (immediate/delayed). Twelve dental implants (2 implants for each dog) were placed immediately and the other 12 dental implants (2 implants for each dog) were placed two months after extraction. The animals were sacrificed 2 months after implant placement. Histological and histometric analysis were performed.

Results: Four implants (3 from immediate and 1 from delayed placement) failed in the experimental group. There were significant differences in the percentage of bone-to-implant contact and marginal bone volume density between the control and the experimental groups. Both parameters were significantly lower in the experimental group than in the control group (P<0.05). There was a tendency toward more marginal bone loss in the experimental group than the control group.

Conclusion: Immediately placed implants have a higher failure rate than delayed placed implants. Untreated experimental periodontitis was correlated with a compromised osseo-integration in delayed placed implants.

Key words: Periodontitis, dental implants, bone-implant interface, osseointegration

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Introduction

During the last 30 years, dental implants have become a commonly used alternative to conventional partial dentures and fixed bridges.[1,2] Although the success rate of implant treatment is high in general, still the implant failure remains an issue.[1-4] Implant failures types are divided into early and late, corresponding to before and after the development of osseointegration, respectively. Early failures are frequently associated with problems during surgical procedures and the initial healing phase.[5-7] Remaining soft tissue between the implant and bone, contamination of the implant surface, excessive surgical trauma with impaired healing potential, and overloading before the occurrence of osseointegration are all possible causes of these failures. However, the reported incidence of early failure is relatively low, ranging from 0.55 to 1.5%.[8,9] Late failures are usually related with problems during restorative procedures or the maintenance phase.[4-8] Late failures mainly result from breakdown of peri-implant alveolar bone (peri-implantitis) due to an imbalanced host response to infection.

Peri-implantitis is a clinical term describing a ‘pathologic process’ affecting the surrounding tissue around an osseointegrated implant in

function.[10-12] The consensus report from the European Workshop on Periodontology defined the term peri-implantitis as a clinical diagnosis that requires the assessment of inflammation in the peri-implant tissue as well loss of supporting bone.[13] As the number of patients treated with dental implants increases, the incidence of peri-implantitis will also increase, causing a significant future problem in health care.

DeBoever & DeBoever reported that following implant insertions in patients with residual dentition, the surfaces of implants were colonized by the same bacteria present on the surfaces of the remaining teeth.[14] Consequently, it has been debated whether a history of periodontitis in patients restored with dental implant increases the risk of peri-implantitis complications.

A recent retrospective study demonstrated that early failure was associated with a history of severe periodontal disease.[15] Peri-implant disease shares various risk factors with periodontitis and also has a similar pathologic mechanism.[16] Although some investigators have reported favorable implant survival rates in periodontally compromised patients,[17,18] previous reviews have showed strong evidence that poor oral hygiene and a history of periodontitis are risk factors for peri-implant disease.[19,20] A history of chronic periodontitis was correlated with higher failure rates, greater marginal bone loss, and more biological complications in dental implants.[20,21] However, these

studies focused on the history of periodontitis, not on active periodontitis, and on long-term prognosis, not on early wound healing around dental implants or osseointegration.

Immediate implant placement was first introduced by Schulte & Heimke.[22] Later histologic studies confirmed that it was a successful procedure.[23,24] A retrospective clinical study demonstrated that the procedure did not affect the long-term survival rate of dental implants.[25] Immediate placement has since become a routine procedure, but it is contraindicated at infected sites with periapical and periodontal lesions.[26-28] On the other hand, in animal studies, successful osseointegration of immediately placed implants have been reported at sites with periapical lesions[29] and periodontal lesions.[30]

Bacterial colonization at newly placed implants occurs rapidly. The concept that microorganisms are indispensable for the development of infections around dental implants is well supported by the many studies.[31,32] The microorganisms associated with peri-implantitis are reported as being similar to that associated with periodontitis and it has been suggested that periodontal pockets of remaining teeth may act as a reservoir for microorganisms to colonize newly placed implants.[32]

In partially edentulous patients, oral ecological conditions influencing the biofilm on the surface of dental implants might vary from those in fully

edentulous patients.[33] Thus, gingivitis or periodontitis of the residual dentition could have an effect on peri-implant tissue health. Periodontal treatment of the residual dentition was recommended prior to implant surgery as a routine part of treatment planning.[34,35] Currently, limited information is available concerning how untreated periodontitis of the residual dentition affects wound healing around dental implants. Therefore, the primary purpose of the present experiment was to study healing around dental implants adjacent to teeth with untreated experimental periodontitis. The second purpose was to compare how immediately placed implants and implants placed on the healed ridge are affected by remaining periodontitis.

Materials and Methods

Four groups were divided according to presence of periodontitis adjacent to the edentulous area and the timing of implant placement. The groups were named the EI, ED, CI, and CD groups (Table 1):

1)experimental periodontitis/immediate placement (EI); 2)experimental periodontitis/delayed placement (ED); 3)control/immediate placement (CI); 4)control/ delayed placement (CD)

Animals

This study was approved by the Seoul National University Institutional Animal Care and Use Committee at Seoul National University, Seoul, Korea (SNU-130806-4-1).

Six male beagle dogs, 1–2 years old and weighing 10–12 kg, were used in this study. All of the dogs had fully erupted permanent dentition. During the experiment, the dogs were housed individually under ambient temperatures of 20–25□, relative humidity of 30–70%, and were fed a soft diet. All clinical and surgical procedures were performed under general anesthesia with intravenous sodium pentobarbital (30 mg/kg)[†]. Local anesthesia was also

provided using 2% lidocaine hydrochloride with 1:100,000 epinephrine‡ at the surgical sites.

Experimental periodontitis

Experimental periodontitis was induced as follows. Using ligature wires and retraction cords, experimental periodontitis was induced in 3 beagle dogs in the experimental group. Retraction cords were soaked in a suspension of *Porphyromonas gingivalis* ATCC 33277 and wound around the ligature wires. Wires were tied to the cervical area of the mandibular first and third premolars (P_1, P_3) and first molars (M_1). The wires and retraction cords were checked routinely, and all remained in the position. After 3 months, experimental periodontitis was induced (Fig. 1B) and alveolar bone loss was identified via periapical radiography (Fig. 1D). Approximately 1-2 mm of alveolar bone loss was observed at the interproximal area in the radiographs. Clinical attachment loss was 1-3 mm at the buccal and lingual area with or without furcation involvement. Inflamed gingiva and supra/subgingival calculus were also found in the clinical examination. Increased mobility of teeth was not observed. In the other 3 dogs (the control group), scaling and plaque control procedures were performed once a month to prevent gingival inflammation. The healthy condition of the periodontium was confirmed clinically (Fig. 1A) and

radiographically (Fig. 1C).

Surgical procedure

All of the procedures followed the study design and experimental process (Fig. 2). In the delayed implant placement groups (ED, CD), mandibular second premolars and fourth premolars were extracted at baseline. The teeth were sectioned in the buccolingual direction at the bifurcation area using a high-speed handpiece and a diamond point so that the roots could be individually extracted without damaging the bony walls. After 2 months of healing, 12 dental implants were installed in the ED and CD groups.

In the immediate placement groups (EI, CI), the same extraction procedures were performed 2 months from baseline. Twelve dental implants were placed immediately after extraction. The mesiodistal position of the immediately-placed implants was the center of the extraction socket of the distal roots of the second premolar (P2) and the mesial roots of the fourth premolar (P4). The platform for the implants was positioned at the level of the buccal bone crest (Fig. 3). In the experimental group, the ligatures were removed after implant installation. The fixtures used in the present study had a diameter of 3.4 mm, a length of 10 mm and had been sandblasted with large grit and acid etched with Ra 1.2 μm[§]. The animals

were sacrificed 2 months after implant installation, 4 months from baseline.

Histological examination and histometric analysis

On the scheduled date, the animals were anesthetized and humanely euthanized. The mandible was surgically removed from each animal. Twenty-four tissue blocks, each containing one implant and the surrounding tissue, were prepared. Specimens were immediately placed in 10% neutral-buffered formalin for 48 hours. After tissue fixation, the tissue blocks were trimmed to a suitable size and then undecalcified and embedded in polymethyl-methacrylate. Each block was sectioned parallel to the implant axis. Sections were ground to approximately 50 μm in thickness with a microgrinding unit. Two sections near the center of the fixture were used for histometric analysis of each implant. One section was stained with hematoxylin-eosin and the other was stained with Masson-Goldner trichrome for detailed analysis of new bone.

Using an optical microscope, histological analysis was conducted at x12 magnification. At x40 magnification, the images of the slides captured by a digital camera were used for histometric analysis.

The data obtained from the buccal and lingual sides of each implant were

as follows: (1) the percentage of bone-to-implant contact (BIC %) from the first bone-to-implant contact point (fBIC) to the bottom of the implant; (2) the percentage of mineralized tissue around the implant in region of coronal 3 mm; (3) marginal bone loss (IS-fBIC, mm) from the implant shoulder (IS) to the most coronal bone-to-implant level (fBIC); (4) reduced ridge height (IS-RC, mm) from the implant shoulder (IS) to the alveolar ridge crest (RC) (Fig. 4A).

The BIC and dimensions of alveolar bone loss (IS-fBIC, IS-RC) was measured with the aid of an image processing program[¶].

The percentage of mineralized tissue was evaluated near the implant surface, in the region of interest between the pitch of the threads (Fig. 4B).[36] Only the marginal 3 mm was used to investigate the quality of marginal bone. Each slide was painted using software[#]. The number of pixels corresponding to mineralized and non-mineralized tissue was counted using software[**].

Statistical analysis

For each group, mean values and standard deviation (SD) were calculated, and descriptive analysis was performed with software[††]. For the present study, six implants were used in each group. The Mann-Whitney U test was chosen to analyze differences between groups because a normal distribution could not

be assumed in this sample size. The results of all comparisons were reported at the 0.05 significance level.

Results

Clinical findings

All surgical sites in the control group showed uneventful healing without significant signs of inflammation, while 4 (3 from the immediate, 1 from the delayed implant placement group) of 12 implants in the experimental group failed without osseointegration at different time points after implant placement.

Histological analysis

Immediate implant placement groups [EI (Fig 5D), CI (Fig5C)] showed greater marginal bone loss than the corresponding delayed groups [ED (Fig 5B), CD (Fig 5A)]. In the immediate placement groups (EI, CI), the bone-to-implant contact did not seem as close as that of the delayed implant placement groups (ED, CD). In the EI group, marginal bone loss extending to the second macro-thread was found on the buccal and lingual side. Even in the CI group marginal bone loss extending to the end of the micro-thread was apparent when compared with the CD group. An irregular cortical bone surface was mainly observed in the EI group, but was also found partly on the buccal side of the CI group.

The delayed placement group (ED, CD) showed generally well-healed alveolar bone around the dental implant, and did not show any severe

horizontal or vertical marginal bone loss. Cortical bone was well rounded and contacted implant surface without any irregularities. A higher percentage of bone-to-implant contact was found compared to the experimental groups (EI, ED) with periodontitis.

Histometric analysis

The percentage of bone-to-implant contact (BIC) around the whole length of the implants is shown in Table 1. The mean BIC was significantly higher at 75.4±8.3% in the CD group compared to 49.9±17.3% in the ED group ($p<0.05$).

Marginal bone volume density (BVD), the percentage of mineralized tissue, was calculated as shown in Table 1. Due to the three failed implants in the EI group and some artifacts in the slides of the EI group, data for BVD of the EI group were insufficient for statistical analysis. The BVD of the CD group was significantly greater than that of the ED group on the buccal side (88±7% vs. 55±9%, respectively; $p<0.05$, Table 1). The marginal bone volume density of the CD group on the lingual side was also higher than that of the ED group (68±31% vs. 45±7%, respectively; Table 1), but this difference did not reach statistical significance.

With the exception of the lingual side of the delayed placement groups

(CD, ED), there was a tendency toward greater bone loss in the experimental periodontitis groups (ED, EI) compared to the corresponding control groups (CD, CI). Immediate placement groups (CI, EI) also had a tendency toward greater bone loss than the corresponding delayed placement groups (CD, ED). Only IS-fBIC on the lingual side in the delayed placement groups showed statistical significance ($p<0.05$); the other differences were not statistically significant (Table 1).

Discussion

An unexpectedly high failure rate (33.3%) was found in the experimental group (ED, EI), while none of implants in the control group (CD, CI) failed. These data could not be analyzed statistically because there was no failure in the control group. However, it is likely that the early failures were affected by periodontitis in the adjacent area considering the reported incidence of early implant failure.[8,9]

Obvious differences exist between chronic and experimentally induced periodontitis, including but not limited to differences in the time required to induce periodontitis, causative factors, and clinical aspects.[37] However, the natural periodontitis of dogs produces inconsistent periodontal lesions with an uneven extent and localization of periodontal inflammation. Therefore, an experimental periodontitis model was proposed to standardize the defects even if there is difference between chronic and experimentally induced periodontitis.

Immediate placement of implants is not recommended in the presence of chronic infections such as periapical lesions and periodontal disease,[27] but a series of histologic studies also showed that successful osseointegration could occur at infected sites.[29,30,38,39] In contrast, implants in the EI group showed a higher failure rate than those in the ED group in the present study.

Mean BIC varied from 49.9 to 75.4% in accordance with previous studies.[24,38,39] Implants in the CD group had significantly greater BIC than those in the ED group, respectively. Data from the EI group could not be used for this analysis due to the unexpected failure of 3 implants. This result can be interpreted in the same context as the failure rate. Remaining periodontal infection could affect the osseointegration of implants in the adjacent area. Between the CD and CI and between the ED and EI groups, mean BIC was not significantly different.

All measurements were performed on bucco-lingually. Although sectioning in a mesio-distal direction may have provided better outcomes, sectioning the center of 2 implants with adjacent teeth proved very difficult. With regard to radiographic measurements, analysis was restricted to confirming that experimental periodontitis had been induced. The present study relied more on histologic and histometric analyses.

Mean marginal bone volume density was also higher around the implants of the control groups compared to the experimental groups. In addition, there was a tendency toward greater marginal bone loss in the experimental groups, although this difference did not reach statistical significance. It seemed that experimental periodontitis affected the quality of the marginal bone and marginal bone loss. There was also a tendency toward greater bone loss in the

CI and EI groups compared to the CD and ED groups, respectively. In this experiment, the implant platform was positioned at the level of the buccal bone crest in all groups. Ridge alteration after extraction might have occurred before implantation in the delayed placement groups, while it began after implant placement in the immediate placement groups. Therefore, marginal bone loss could be exaggerated in the immediate placement groups. In this study, the bucco-lingual position of the implant could not be standardized; the width of the buccal bone and the gap between the implant surface and the buccal wall could not be controlled either. These factors might have affected buccal bone loss.

One possible explanation for implant failure in the ED and EI groups was contamination of the implant surface. Meticulous debridement of the extraction sockets was performed before preparing the implant bed, but some sources of infection still remained, including biofilm and periodontal lesions on the adjacent natural teeth. Without periodontal treatment, saline irrigation during drilling could transport pathogens to the implant surface.

A number of studies[29,30,38,39] with designs similar to the present study reported successful osseointegration at infected sites in dogs. The investigators, however, extracted all premolars on which periodontitis was induced prior to implantation. In the present study, teeth adjacent to the implants remained in

place, with unresolved tissue inflammation and pre-surgical antibiotics were not provided; this could explain why our results differ from those of previous studies.

Initial discussion with IRB forecasted the chosen sample size to be large enough to be able to conduct statistical analyses and draw sound conclusions based on previous studies with similar experimental designs. However, unexpected implant failure occurred in experimental group, it becomes difficult to extrapolate the results obtained to have any significance. It was not anticipated because there was not any previous study with untreated experimental periodontitis as we tried in the present study. It can be assumed that this failure was also influenced either directly or indirectly by the presence of experimental periodontitis. Using the present investigation as a pilot study, further research into the relationship between osseointegration of dental implants and existing periodontitis using a greater sample size is required.

Within the limitations of this study, it was found that early wound healing or osseointegration around dental implants could be disturbed by induced periodontitis in adjacent teeth. Immediate implant placement led to a greater risk of this than delayed implant placement. Thus, periodontal inflammation of the remaining dentition should be resolved prior to implant placement. In the

present study, we confirmed the importance of the periodontal condition of adjacent teeth and the timing of implant placement.

Conclusion

Immediately placed implants have a higher failure rate than delayed placed implants. Untreated experimental periodontitis was correlated with a compromised osseointegration in delayed placed implants.

Footnotes

†Hanlim Pharm., Seoul**,** Korea

‡Huons, Sungnam, Korea

§Implantium, Dentium, Seoul, Korea

Olympus BH-2; Olympus Optical, Osaka, Japan

¶ImageJ, National Institute of Health, USA

#Adobe Photoshop CS6, Mountain View, CA, USA

**ScopeEye 3.6, TOMORO, Samkyung, Seoul, Korea

††PASW 18, IBM, NY, USA

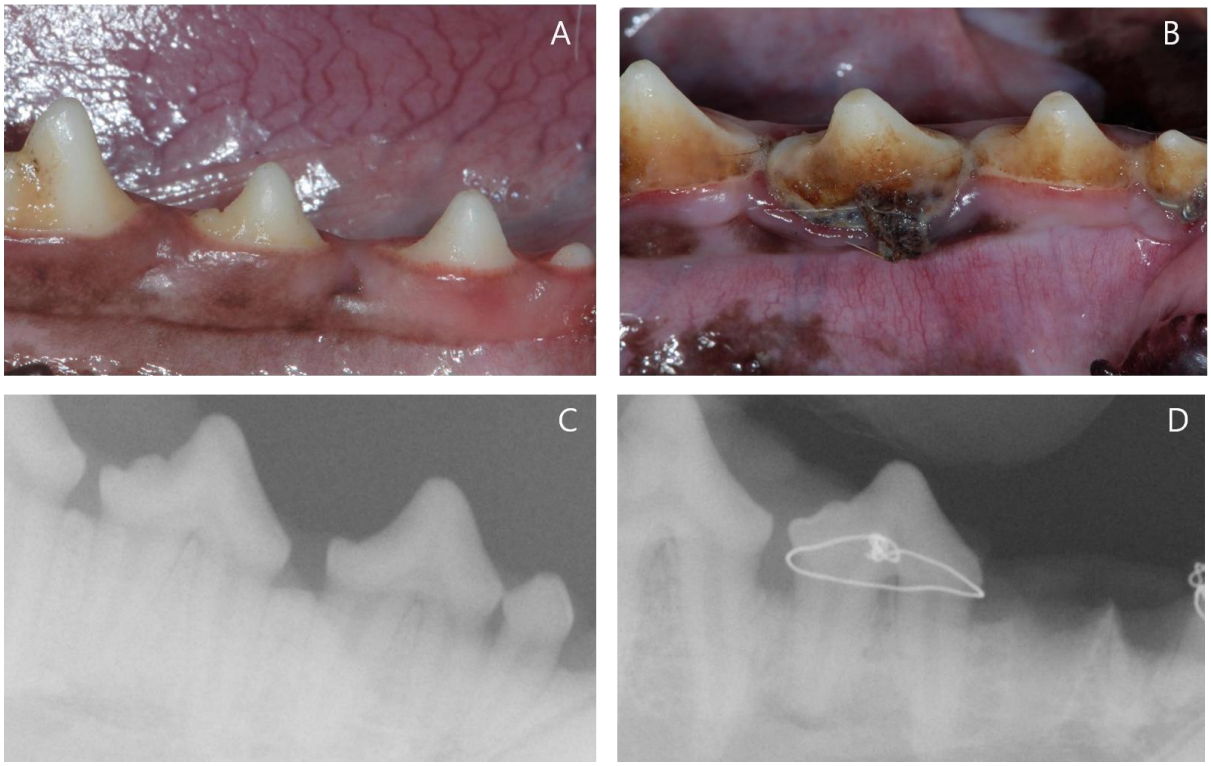

Figure 1. Clinical photos and radiographs taken before and after induction of experimental periodontitis. Healthy periodontal tissue (A) became inflamed (B) due to subgingivally-placed wires wound with ligature soaked in *Porphyromonas gingivalis*. Periapical radiographs of the site confirmed the presence of healthy periodontal tissue (C) and experimental periodontitis (D).

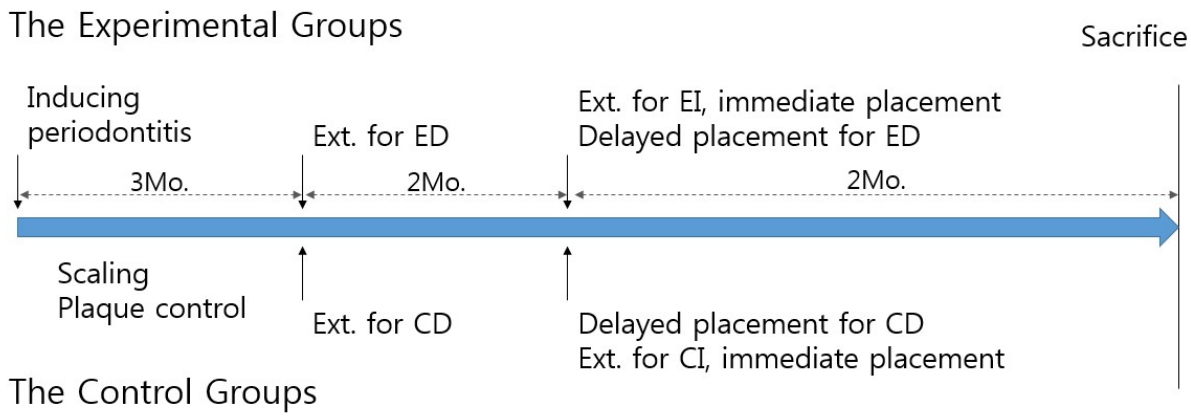

Figure 2. Description of the experimental process. Experimental subgroups: EI (n=6), experimental periodontitis + immediate implant placement after extraction; ED (n=6), experimental periodontitis + delayed implant placement 2 months after extraction; Control subgroups: CI (n=6), healthy periodontium + immediate implant placement; CD (n=6), healthy periodontium + delayed implant placement 2 months after extraction. In the experimental group (EI, ED), it took 3 months to induce periodontitis. In the control group (CI, CD), scaling and plaque control procedures were performed once a month for 3 months. All animals were sacrificed 2 months after implant placement.

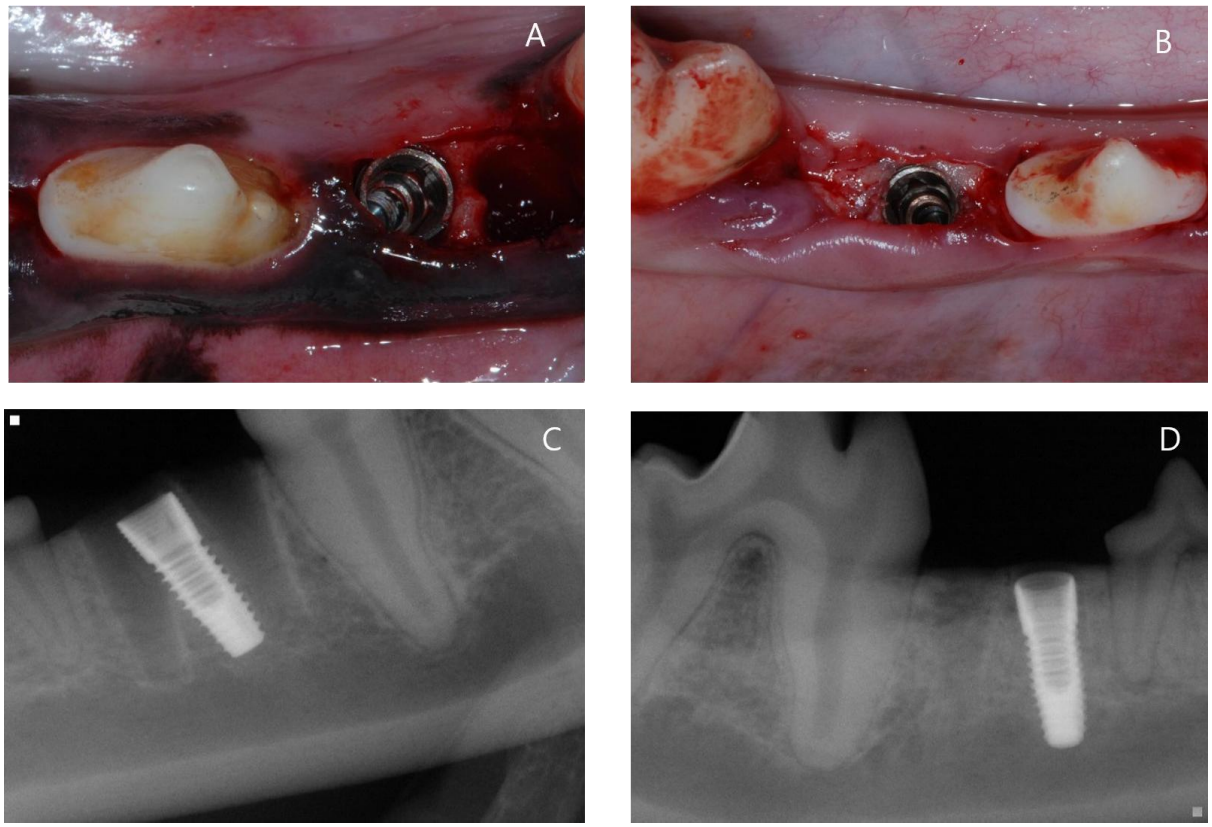

Figure 3. Clinical photos of immediate implant placement (A) and implant placement 2 months after extraction (B). Corresponding periapical radiographs of immediate implant placement (C) and implant placement 2 months after extraction (D).

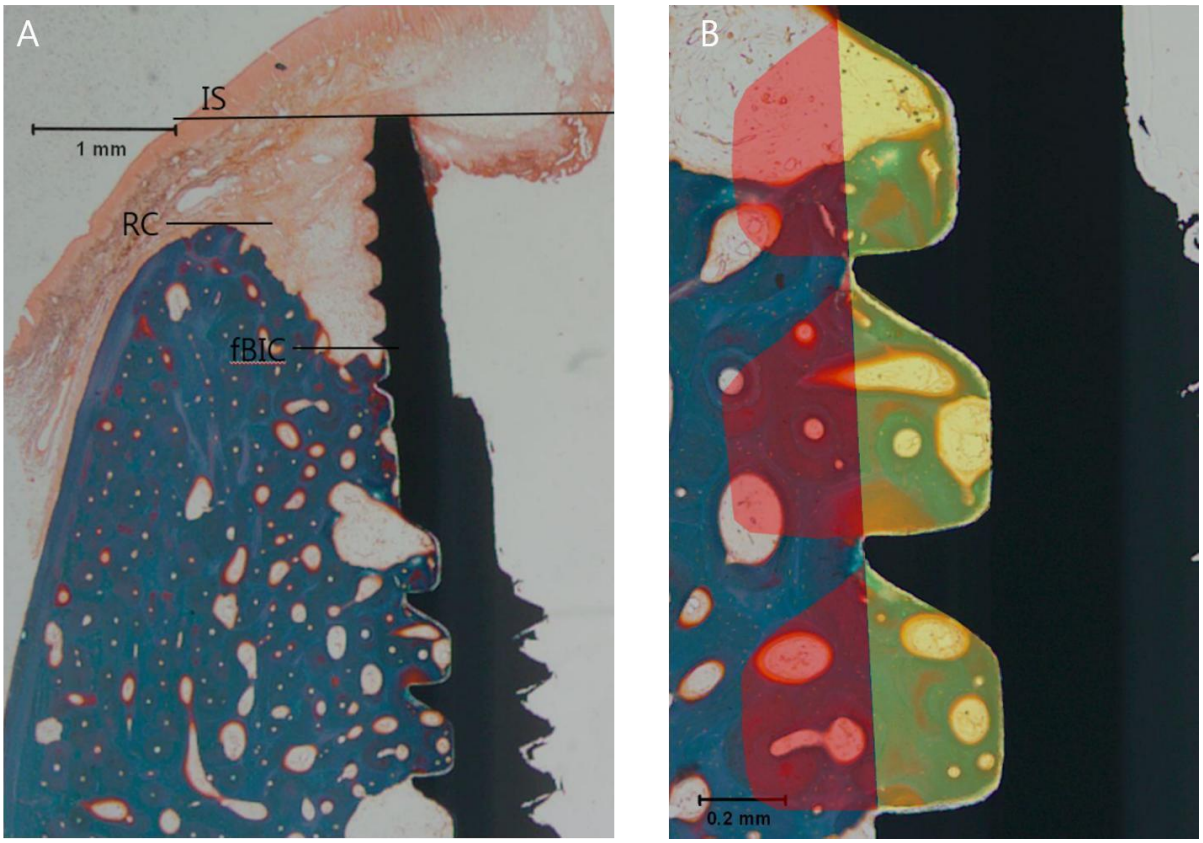

Figure 4. Histometric data. Implant shoulder (IS), ridge crest (RC), and the most coronal bone-to-implant contact (fBIC) were chosen as references for marginal bone loss (A) (x12 magnification). The area between implant threads and the reproduced mirror area were selected as areas of interest (B) (x40 magnification).

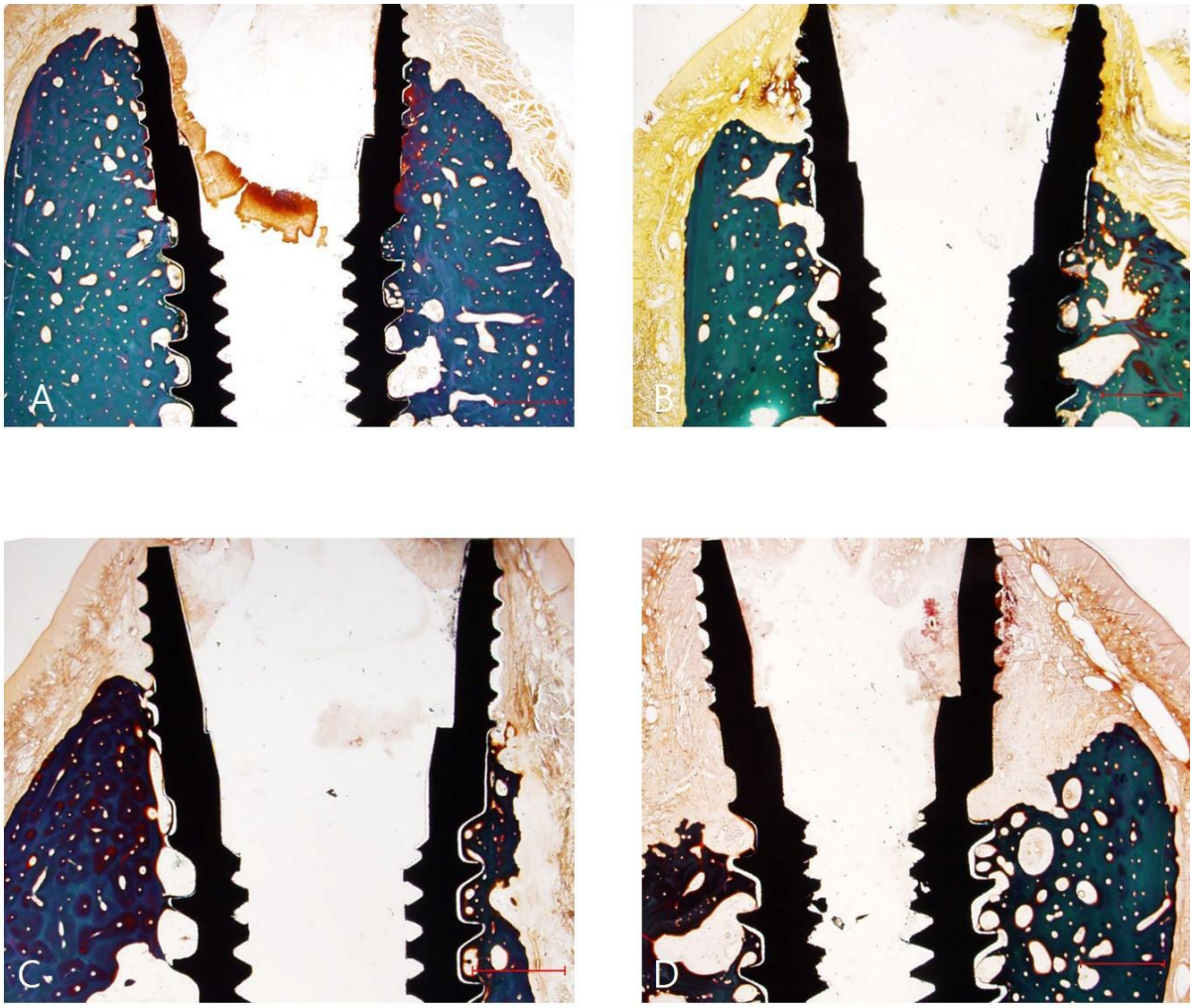

Figure 5. Representative microscopic images of the CD group (A), ED group (B), CI group (C), and EI group (D). The scale bar is 0.5 mm in length (x12 magnification).

Immediate placement groups(Fig. 5C, 5D) showed greater marginal bone loss than the delayed groups(Fig. 5A, 5B). In the EI groups (Fig. 5D), marginal bone loss extending to the second macrothread was found on the buccal and lingual sides.

A higher percentage of BIC in control groups(Fig. 5A, 5C) was found compared with experimental groups(Fig. 5B, 5D) with periodontitis.

Table1. Bone-to-implant contact ratio and marginal bone volume density

	Group			
	CD (n=6)	ED (n=5)	CI (n=6)	EI (n=3)
BIC (%)	75.4±8.3*	49.9±17.3*	72.2±12.2	63.0±10.4
Marginal bone volume density (%)				
Buccal	88±7†	55±9†	67±18	42
Lingual	68±31	45±7	76±23	21
Marginal bone level (mm)				
IS-fBIC				
Buccal	2.02±1.01	3.11±0.89	2.24±0.49	3.59±2.00
Lingual	1.20±0.41‡	3.20±1.13‡	1.67±0.45	3.45±2.43
IS-RC				
Buccal	1.90±0.93	2.59±0.99	2.22±0.53	3.39±1.69
Lingual	0.79±0.44	0.40±1.20	1.55±0.60	2.19±0.77

Statistical analysis was performed using Mann-Whitney U test.

*, †, and ‡ indicate statistical significance ($p<0.05$).

The mean BIC was significantly higher in the CD group compared with in the ED group ($p<0.05$). The BVD of the CD group was significantly greater than that of the ED group on the buccal side. The marginal BVD of the CD group on the lingual side was also higher than that of the ED group, but this difference did not reach statistical significance.

References

1. Adell R, Eriksson B, Lekholm U, Brånemark PI, Jemt T. (1990) Long-term follow-up study of osseointegrated implants in the treatment of totally edentulous jaws. *Int J Oral Maxillofac Implants*. Winter;5(4): 347-59.

2. Esposito M, Ardebili Y, Worthington HV. (2014) Interventions for replacing missing teeth: different types of dental implants. *Cochrane Database Syst Rev.* Jul 22;7.

3. el Askary AS, Meffert RM, Griffin T. (1999) Why do dental implants fail? Part I. *Implant Dent.*;8(2):173-85.

4. el Askary AS, Meffert RM, Griffin T. Why do dental implants fail? Part II. *Implant Dent*. 1999;8(3):265-77.

5. Esposito M, Hirsch JM, Lekholm U, Thomsen P. (1998) Biological factors contributing to failures of osseointegrated oral implants. (II). Etiopathogenesis. *Eur J Oral Sci.* Jun;106(3):721-64.

6. Manor Y, Oubaid S, Mardinger O, Chaushu G, Nissan J. (2009) Characteristics of early versus late implant failure: a retrospective study. *J Oral Maxillofac Surg.* Dec;67(12):2649-52.

7. Esposito M, Lausmaa J, Hirsch JM, Thomsen P. (1999) Surface analysis

of failed oral titanium implants. *J Biomed Mater Res.*;48(4):559-68.

8. Buser D, Mericske-Stern R, Bernard JP, Behneke A, Behneke N, Hirt HP, Belser UC, Lang NP. (1997) Long-term evaluation of non-submerged ITI implants. Part 1: 8-year life table analysis of a prospective multi-center study with 2359 implants. *Clin Oral Implants Res.* Jun;8(3):161-72.

9. Friberg B, Jemt T, Lekholm U. (1991) Early failures in 4,641 consecutively placed Brånemark dental implants: a study from stage 1 surgery to the connection of completed prostheses. *Int J Oral Maxillofac Implants.* Summer;6(2):142-6.

10. Furst, M., Salvi, G. E., Lang, N. P. & Persson, G. R. (2007) Bacterial colonization immediately after installation on oral titanium implants. *Clin Oral Implants Res.* Aug;18(4):501–508.

11. Salvi, G. E., Fu¨rst, M. M., Lang, N. P. & Persson, G. R. (2008) One-year bacterial colonization patterns of Staphylococcus aureus and other bacteria at implants and adjacent teeth. *Clin Oral Implants Res.* Mar;19(3):242–248.

12. Karoussis, I.K., Muller, S., Salvi, G.E., Heitz-Mayfield, L.J.A., Bragger, U. & Lang, N.P. (2004) Association between periodontal and peri-implant conditions: a 10-year prospective study. *Clin Oral*

Implants Res. Feb;15(1):1-7.

13. Albrektsson, T. & Isidor, F. (1994) Consensus report session IV. In: Lang, N.P. & Karring, T., eds. *Proceedings of the First European Workshop on Periodontology*, 365–369.

14. De Boever, A.L. & De Boever, J.A. (2006) Early colonization of non-submerged dental implants in patients with a history of advanced aggressive periodontitis. *Clin. Oral Implants Res.* Feb;17(1):8-17.

15. Olmedo-Gaya MV, Manzano-Moreno FJ, Cañaveral-Cavero E, de Dios Luna-Del Castillo J, Vallecillo-Capilla M. (2016) Risk factors associated with early implant failure: A 5-year retrospective clinical study. *J Prosthet Dent.* Feb;115(2):150-5.

16. Heitz-Mayfield LJ, Lang NP. (2010) biology of chronic and aggressive periodontitis vs. peri-implantitis. *Periodontol 2000.* Jun;53:167-81.

17. Baelum V, Ellegaard B. (2004) Implant survival in periodontally compromised patients. *J Periodontol*. Oct;75(10):1404-12.

18. Ellegaard B, Baelum V, Karring T. (1997) Implant therapy in periodontally compromised patients. *Clin Oral Implants Res.* Jun;8(3):180-8.

19. Heitz-Mayfield LJ. (2008) Peri-implant diseases: diagnosis and risk indicators. *J Clin Periodontol.* Sep;35(8 Suppl):292-304.

20. Safii SH, Palmer RM, Wilson RF. (2010) Risk of implant failure and marginal bone loss in subjects with a history of periodontitis: a systematic review and meta-analysis. *Clin Implant Dent Relat Res.* Sep;12(3):165-74.

21. Karoussis IK, Salvi GE, Heitz-Mayfield LJ, Brägger U, Hämmerle CH, Lang NP. (2003) Long-term implant prognosis in patients with and without a history of chronic periodontitis: a 10-year prospective cohort study of the ITI Dental Implant System. *Clin Oral Implants Res.* Jun;14(3):329-39.

22. Schulte W, Heimke G. (1976) The Tubinger immediate implant. *Die Quintessenz* 27.

23. Anneroth G, Hedström KG, Kjellman O, Köndell PA, Nordenram A. (1985) Endosseus titanium implants in extraction sockets. An experimental study in monkeys. *Int J Oral Surg.* 1985 Feb;14(1):50-4.

24. Barzilay I, Graser GN, Iranpour B, Natiella JR. (1991) Immediate implantation of a pure titanium implant into an extraction socket: report of a pilot procedure. I*nt J Oral Maxillofac Implants.* Fall;6(3):277-84.

25. Evian CI, Emling R, Rosenberg ES, Waasdorp JA, Halpern W, Shah S, Garcia M. (2004) Retrospective analysis of implant survival and the influence of periodontal disease and immediate placement on long-term

results. *Int J Oral Maxillofac Implants.* May-Jun;19(3):393-8.

26. Barzilay I. (1993) Immediate implants: their current status. *Int J Prosthodont.* Mar-Apr;6(2):169-75.

27. Becker W, Becker BE. (1990) Guided tissue regeneration for implants placed into extraction sockets and for implant dehiscences: surgical techniques and case report. *Int J Periodontics Restorative Dent.*;10(5):376-91.

28. Werbitt MJ, Goldberg PV. (1992) The immediate implant: bone preservation and bone regeneration. *Int J Periodontics Restorative Dent.*;12(3):206-17.

29. Novaes AB Jr, Vidigal Júnior GM, Novaes AB, Grisi MF, Polloni S, Rosa A. (1998) Immediate implants placed into infected sites: a histomorphometric study in dogs. *Int J Oral Maxillofac Implants.* May-Jun;13(3):422-7.

30. Novaes AB Jr, Marcaccini AM, Souza SL, Taba M Jr, Grisi MF. (2003) Immediate placement of implants into periodontally infected sites in dogs: a histomorphometric study of bone-implant contact. *Int J Oral Maxillofac Implants.* May-Jun;18(3):391-8.

31. Quirynen, M., De Soete, M. & van Steenberghe, D. (2002) Infectious risks for oral implants: a review of the literature. *Clin Oral Implants*

Res 13, 1–19.

32. Quirynen, M., Vogels, R., Peeters, W., van Steenberghe, D., Naert, I. & Haffajee, A. (2006) Dynamics of initial subgingival colonization of 'pristine' peri-implant pockets. *Clin. Oral Implants Res.* Feb;17(1):25-37.

33. Mombelli A, Marxer M, Gaberthüel T, Grunder U, Lang NP. (1995) The microbiota of osseointegrated implants in patients with a history of periodontal disease. *J Clin Periodontol.* Feb;22(2):124-30.

34. Brägger U, Bürgin WB, Hämmerle CH, Lang NP. (1997) Associations between clinical parameters assessed around implants and teeth. *Clin Oral Implants Res.* Oct;8(5):412-21.

35. Brägger U, Hämmerle C, Weber HP. (1990) Fixed reconstructions in partially edentulous patients using two-part ITI implants (Bonefit) as abutments. *Clin Oral Implants Res.* Dec;1(1):41-9.

36. Vignoletti F, Johansson C, Albrektsson T, De Sanctis M, San Roman F, Sanz M. (2009) Early healing of implants placed into fresh extraction sockets: an experimental study in the beagle dog. De novo bone formation. *J Clin Periodontol.* Mar;36(3):265-77.

37. Haney JM, Zimmerman GJ, Wikesjo (1995) UM. Periodontal repair in dogs: evaluation of the natural disease model. *J Clin*

Periodontol;22:208–213.

38. Novaes AB Jr, Souza SL, de Oliveira PT, Souza AM. (2002) Histomorphometric analysis of the bone-implant contact obtained with 4 different implant surface treatments placed side by side in the dog mandible. *Int J Oral Maxillofac Implants.* May-Jun;17(3):377-83.

39. Novaes AB Jr, Papalexiou V, Grisi MF, Souza SS, Taba M Jr, Kajiwara JK. (2004) Influence of implant microstructure on the osseointegration of immediate implants placed in periodontally infected sites. A histomorphometric study in dogs. *Clin Oral Implants Res.* Feb;15(1):34-43.

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- 국문초록 -

치료되지 않은 치주염이 치과 임플란트 의 골유착에 미치는 영향

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치주염과 임플란트 주위염 사이의 관계에 대한 이전의 많은 연구들이 있었지만 치주염이 자연치의 옆에 새롭게 심겨진 치과 임플란트의 치유와 골유착에 어떻게 영향을 미치는 지에 대해서는 제한적인 정보만이 알려져 있다. 본 연구의 목적은 실험적으로 유발된 치료되지 않은 치주염에 인접한 치과 임플란트의 치유과정을 평가하는 것이다.

본 연구는 6마리의 수컷 비글견을 대상으로 하였다. 3마리(대조군)는 치석제거와 치태조절 과정을 거쳤고, 다른 3마리(실험군)는 압배사와 결찰용철사를 각각의 소구치와 1대구치의 치은 연하부에 넣었다. 실험적으로 유발된 치주염을 3개월이 지나서 확인하였다. 각각의 대조군이나 실험군은 임플란트의 식립시기(발치 즉시 / 지연 식립)에 따라서 2개의 군으로 추가 분류하였다. 12개의 치과 임플란트(각각의 개에 2개씩)를 발치 후 즉시 식립하였고 다른 12개의 치과 임플란트(역시 각각의 개에 2개씩)는 발치 후 2개월 후에 식립하였다.

실험군에 속한 4개의 임플란트(3개는 즉시 식립, 1개는 지연식립)가 실패하였다. 대조군과 실험군 사이에 골-임플란트 접촉면적과 주변골의 밀도에서 현저한 차이를 볼 수 있었다. 두 지표 모두 대조군보다 실험군에서 현저히 낮았다 ($P<0.05$). 주변 골소실의 정도도 대조군보다 실험군에서 더 심한 경향이 있었다.

결론적으로 실험군에서 즉시 식립을 한 임플란트의 경우는 지연식립을 한 임플란트의 경우보다 더 높은 실패율을 보였고, 치료되지 않은 실험적인 치주염은 지연 식립을 한 임플란트의 골유착 저하와 관계가 있었다.

주요어 : 치주염, 치과 임플란트, 골-임플란트 경계면, 골유착

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